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NEWS	1		Web Page for STN Seminar Schedule - N. America
NEWS	2	DEC 01	ChemPort single article sales feature unavailable
NEWS	3	APR 03	CAS coverage of exemplified prophetic substances enhanced
NEWS	4	APR 07	STN is raising the limits on saved answers
NEWS	5	APR 24	CA/CAplus now has more comprehensive patent assignee information
NEWS	6	APR 26	USPATFULL and USPAT2 enhanced with patent assignment/reassignment information
NEWS	7	APR 28	CAS patent authority coverage expanded
NEWS	8	APR 28	ENCOMPLIT/ENCOMPLIT2 search fields enhanced
NEWS	9	APR 28	Limits doubled for structure searching in CAS REGISTRY
NEWS	10	MAY 08	STN Express, Version 8.4, now available
NEWS	11	MAY 11	STN on the Web enhanced
NEWS	12	MAY 11	BEILSTEIN substance information now available on STN Easy
NEWS	13	MAY 14	DGENE, PCTGEN and USGENE enhanced with increased limits for exact sequence match searches and introduction of free HIT display format
NEWS	14	MAY 15	INPADOCDB and INPAFAMDB enhanced with Chinese legal status data
NEWS	15	MAY 28	CAS databases on STN enhanced with NANO super role in records back to 1992
NEWS	16	JUN 01	CAS REGISTRY Source of Registration (SR) searching enhanced on STN
NEWS	17	JUN 26	NUTRACEUT and PHARMAML no longer updated
NEWS	18	JUN 29	IMSCOPROFILE now reloaded monthly
NEWS	19	JUN 29	EPFULL adds Simultaneous Left and Right Truncation (SLART) to AB, MCLM, and TI fields
NEWS	20	JUL 09	PATDPAFULL adds Simultaneous Left and Right Truncation (SLART) to AB, CLM, MCLM, and TI fields
NEWS	21	JUL 14	USGENE enhances coverage of patent sequence location (PSL) data
NEWS	22	JUL 27	CA/CAplus enhanced with new citing references
NEWS	23	JUL 16	GBFULL adds patent backfile data to 1855
NEWS	24	JUL 21	USGENE adds bibliographic and sequence information

NEWS EXPRESS MAY 26 09 CURRENT WINDOWS VERSION IS V8.4,
AND CURRENT DISCOVER FILE IS DATED 06 APRIL 2009.

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FILE 'HOME' ENTERED AT 07:40:47 ON 28 JUL 2009

=> file registry

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.22	0.22

FILE 'REGISTRY' ENTERED AT 07:41:08 ON 28 JUL 2009

Property values tagged with IC are from the ZIC/VINITI data file
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STRUCTURE FILE UPDATES: 26 JUL 2009 HIGHEST RN 1169218-44-3
DICTIONARY FILE UPDATES: 26 JUL 2009 HIGHEST RN 1169218-44-3

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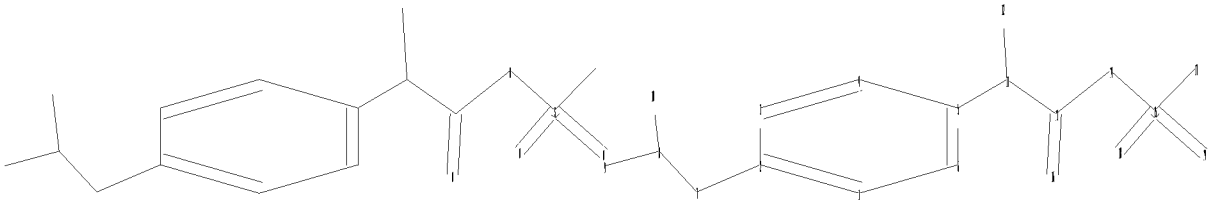
=>
Uploading C:\Program Files\STNEXP\Queries\10588454.str



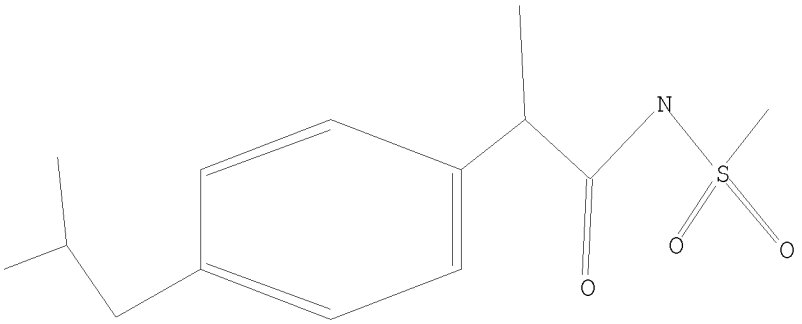
chain nodes :
7 8 9 10 11 12 13 14 15 16 17 18 19
ring nodes :
1 2 3 4 5 6
chain bonds :
2-7 5-11 7-8 8-9 8-10 11-12 11-13 13-14 13-16 14-15 15-17 15-18 15-19

ring bonds :
1-2 1-6 2-3 3-4 4-5 5-6
exact/norm bonds :
13-14 13-16 14-15 15-17 15-18 15-19
exact bonds :
2-7 5-11 7-8 8-9 8-10 11-12 11-13
normalized bonds :
1-2 1-6 2-3 3-4 4-5 5-6

Match level :
1:Atom 2:Atom 3:Atom 4:Atom 5:Atom 6:Atom 7:CLASS 8:CLASS 9:CLASS 10:CLASS
 11:CLASS 12:CLASS 13:CLASS 14:CLASS 15:CLASS 16:CLASS 17:CLASS 18:CLASS
19:CLASS

L1   STRUCTURE UPLOADED

=> d l1
L1 HAS NO ANSWERS
L1   STR



Structure attributes must be viewed using STN Express query preparation.

=> s l1 fam ful
FULL SEARCH INITIATED 07:41:26 FILE 'REGISTRY'
FULL SCREEN SEARCH COMPLETED -   59 TO ITERATE

100.0% PROCESSED   59 ITERATIONS   5 ANSWERS
SEARCH TIME: 00.00.01

L2   5 SEA FAM FUL L1

=> file caplus
COST IN U.S. DOLLARS   SINCE FILE   TOTAL
                                                         ENTRY   SESSION
FULL ESTIMATED COST   73.33   73.55

FILE 'CAPLUS' ENTERED AT 07:41:46 ON 28 JUL 2009
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FILE COVERS 1907 - 28 Jul 2009 VOL 151 ISS 5
FILE LAST UPDATED: 27 Jul 2009 (20090727/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Jun 2009
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Jun 2009

CAplus now includes complete International Patent Classification (IPC) reclassification data for the second quarter of 2009.

CAS Information Use Policies apply and are available at:

http://www.cas.org/legal/infopolicy.html

This file contains CAS Registry Numbers for easy and accurate substance identification.

The ALL, BIB, MAX, and STD display formats in the CA/CAplus family of databases have been updated to include new citing references information. This enhancement may impact record import into database management software. For additional information, refer to NEWS 22.

=> s l2
L3 23 L2

=> s l3 and spin?
746103 SPIN?
L4 3 L3 AND SPIN?

=> d l4 ibib abs 1-3

L4 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2008:412188 CAPLUS
DOCUMENT NUMBER: 148:394429
TITLE: CXC chemokine-mediated signaling targeting for treatment of a myelin disorder
INVENTOR(S): Miller, Robert H.; Padovani-Claudio, Dolly A.
PATENT ASSIGNEE(S): Case Western Reserve University, USA
SOURCE: PCT Int. Appl., 85pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2008039876	A1	20080403	WO 2007-US79602	20070926

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

CA 2664359	A1	20080403	CA 2007-2664359	20070926
US 20090041753	A1	20090212	US 2007-904634	20070926
EP 2066335	A1	20090610	EP 2007-843271	20070926

R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, RS

PRIORITY APPLN. INFO.: US 2006-847656P P 20060926
WO 2007-US79602 W 20070926

AB The invention discloses compns. and methods for targeting CXC chemokine-mediated signaling for treatment of a myelin disorder. The methodol. of the invention can be used to ameliorate neuropathies.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2007:976827 CAPLUS
DOCUMENT NUMBER: 147:314799
TITLE: Reparixin, an inhibitor of CXCR2 function, attenuates inflammatory responses and promotes recovery of function after traumatic lesion to the spinal cord
AUTHOR(S): Gorio, Alfredo; Madaschi, Laura; Zadra, Giorgia; Marfia, Giovanni; Cavalieri, Barbara; Bertini, Riccardo; Di Giulio, Anna Maria
CORPORATE SOURCE: Pharmacological Laboratories, Department of Medicine, Surgery and Dentistry, Faculty of Medicine, University of Milan, Milan, Italy
SOURCE: Journal of Pharmacology and Experimental Therapeutics (2007), 322(3), 973-981
CODEN: JPETAB; ISSN: 0022-3565
PUBLISHER: American Society for Pharmacology and Experimental Therapeutics
DOCUMENT TYPE: Journal
LANGUAGE: English

AB It has been shown that the blockade of CXCR1 and CXCR2 receptors prevents ischemia/reperfusion damage in several types of vascular beds. Reparixin is a recently described inhibitor of human CXCR1/R2 and rat CXCR2 receptor activation. We applied reparixin in rats following traumatic spinal cord injury and determined therapeutic temporal and dosages windows. Treatment with reparixin significantly counteracts secondary degeneration by reducing oligodendrocyte apoptosis, migration to the injury site of neutrophils and ED-1-pos. cells. The observed preservation of the white matter might also be secondary to the enhanced proliferation of NG2-pos. cells. The expression of macrophage-inflammatory protein-2, tumor necrosis factor-α, interleukin (IL)-6, and IL-1β was also counteracted, and the proliferation of glial fibrillary acidic protein-pos. cells was markedly reduced. These effects resulted in a smaller post-traumatic cavity and in a significantly improved recovery of hind limb function. The best beneficial outcome of reparixin treatment required 7-day administration either by i.p. route (15 mg/kg) or s.c. infusion via osmotic pumps (10 mg/kg), reaching a steady blood level of 8 μg/mL. Methylprednisolone was used as a reference drug; such treatment reduced cytokine production but failed to affect the rate of hind limb recovery.

OS.CITING REF COUNT: 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)
REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2006:704377 CAPLUS
DOCUMENT NUMBER: 145:369213
TITLE: Species differences in the pharmacokinetics and metabolism of Reparixin in rat and dog
AUTHOR(S): Midgley, I.; Fitzpatrick, K.; Wright, S. J.; John, B. A.; Peard, A. J.; Major, R. M.; Holding, J. D.; McBurney, A.; Anacardio, R.; Novellini, R.; Ferrari, M. P.
CORPORATE SOURCE: Department of Drug Metabolism, Huntingdon Life Sciences Ltd, Huntingdon, UK
SOURCE: Xenobiotica (2006), 36(5), 419-440
CODEN: XENOBH; ISSN: 0049-8254
PUBLISHER: Taylor & Francis Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The pharmacokinetics and metabolism of Reparixin (formerly Repertaxin), a potent and specific inhibitor of the chemokine CXCL8, were investigated in rats and dogs after i.v. administration of [14C]Reparixin L-lysine salt. Protein binding of Reparixin was investigated in vitro in rat, dog, rabbit, cynomolgus monkey, and human plasma. Plasma protein binding of Reparixin was >99% in the laboratory animals and humans up to 50 μg mL-1, but lower at higher concns. Although radioactivity was rapidly distributed into rat tissues, Vss was low (.apprx.0.15 L kg-1) in both rat and dog. Nevertheless, Reparixin was more rapidly eliminated in rats (t1/2 .apprx.0.5 h) than in dogs (t1/2 .apprx.10 h). Systemic exposure in dog was due primarily to parent drug, but metabolites played a more prominent role in rat. Oxidation of the iso-Bu side-chain was the major metabolic pathway in rat, whereas hydrolysis of the amide bond predominated in dog. Urinary excretion, which accounted for 80-82% of the radioactive dose, was the major route of elimination in both species, and biotransformation of Reparixin was complete before excretion.
OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d l3 1-23 ibib abs

L3 ANSWER 1 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2009:779262 CAPLUS
TITLE: Development and validation of an LC-MS/MS method for determination of methanesulfonamide in human urine
AUTHOR(S): Anacardio, Roberto; Mullins, Frank G. P.; Hannam, Sally; Sheikh, Muhammed S.; O'Shea, Karen; Aramini, Andrea; D'Anniballe, Gaetano; D'Anteo, Loredana; Ferrari, Mauro P.; Allegretti, Marcello
CORPORATE SOURCE: Research Department, Dompe pha.r.ma s.p.a., L'Aquila, Italy
SOURCE: Journal of Chromatography, B: Analytical Technologies in the Biomedical and Life Sciences (2009), 877(22), 2087-2092
CODEN: JCBAAI; ISSN: 1570-0232
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A sensitive and selective liquid chromatog. method coupled with tandem mass spectrometry (LC-MS/MS) was developed and validated for the quantification of methanesulfonamide (MSA) in human urine. MSA is a potential in vivo

metabolite of reparixin, a specific inhibitor of the CXCL8 biol. activity. In this study, a simple derivatization procedure with a new reagent, N-(4-methanesulfonyl-benzoyl)-imidazole, was set up to enable MSA and the internal standard (I.S.), ethanesulfonamide (ESA), to be analyzed by LC-MS/MS. After derivatization, samples were evaporated and reconstituted in 30% acetonitrile, aqueous MSA and I.S. derivs. were separated by reversed phased HPLC (high performance liquid chromatog.) on a Luna 5 μ C18 column and quantitated by MS/MS using electrospray ionization (ESI) and multiple reaction monitoring (MR M) in the neg. ion mode. The most intense [M-H]- MRM transition of derivatized MSA at m/z 276.2 → 197.2 was used for quantitation and the transition at m/z 290.2 → 211.2 was used to monitor derivatized ESA. The method was linear over the concentration range from 1 to 100 μg/mL, with a lower limit of quantitation of 1 μg/mL. The intra- and inter-day precisions were less than 5.5% and 10.1%, resp., and the accuracies were between -4.0% and +11.3%. The method was successfully applied to quantify levels of MSA in human urine after i.v. administration of reparixin to healthy volunteers.

L3 ANSWER 2 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2008:1475435 CAPLUS
DOCUMENT NUMBER: 150:75537
TITLE: Novel Role of CXCR2 in Regulation of γ-Secretase Activity
AUTHOR(S): Bakshi, Pancham; Margenthaler, Elaina; Laporte, Vincent; Crawford, Fiona; Mullan, Michael
CORPORATE SOURCE: Roskamp Institute, Sarasota, FL, 34203, USA
SOURCE: ACS Chemical Biology (2008), 3(12), 777-789
CODEN: ACBCCT; ISSN: 1554-8929
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Alzheimer's disease (AD) is a progressive chronic disorder that leads to cognitive decline. Several studies have associated up-regulation of some of the chemokines and/or their receptors with altered APP processing leading to increased production of β-amyloid protein (Aβ) and AD pathol. changes. However, there is no direct evidence to date to determine whether the altered processing of APP results in up-regulation of these receptors or whether the up-regulation of the chemokine receptors causes modulated processing of APP. In the current study, we demonstrate that treatment of the chemokine receptor CXCR2 with agonists leads to enhancement of Aβ production and treatment with antagonists or immunodepletion of CXCR2's endogenous agonists leads to Aβ inhibition. Further, we found that the inhibitory effect of the antagonist of CXCR2 on Aβ40 and Aβ42 is mediated via γ-secretase, specifically through reduction in expression of presenilin (PS), one of the γ-secretase components. Also, in vivo chronic treatment with a CXCR2 antagonist blocked Aβ40 and Aβ42 production Using small interfering RNAs for CXCR2, we further showed that knockdown of CXCR2 in vitro accumulates γ-secretase substrates C99 and C83 with reduced production of both Aβ40 and Aβ42. Taken together, these findings strongly suggest for the first time that up-regulation of the CXCR2 receptor can be the driving force in increased production of Aβ. Our findings unravel new mechanisms involving the CXCR2 receptor in the pathogenesis of AD and pose it as a potential target for developing novel therapeutics for intervention in this disease. Also, we propose here a new chemical series of interest that can serve as a prototype for drug development.
REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 3 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2008:1167893 CAPLUS
DOCUMENT NUMBER: 149:439943
TITLE: Therapeutic inhibition of CXCR2 by Reparixin attenuates acute lung injury in mice
AUTHOR(S): Zarbock, A.; Allegretti, M.; Ley, K.
CORPORATE SOURCE: Division of Inflammation Biology, La Jolla Institute for Allergy and Immunology, La Jolla, CA, USA
SOURCE: British Journal of Pharmacology (2008), 155(3), 357-364
CODEN: BJPCBM; ISSN: 0007-1188
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Acute lung injury (ALI) remains a major challenge in critical care medicine. Both neutrophils and chemokines have been proposed as key components in the development of ALI. The main chemokine receptor on neutrophils is CXCR2, which regulates neutrophil recruitment and vascular permeability, but no small mol. CXCR2 inhibitor has been demonstrated to be effective in ALI or animal models of ALI. To investigate the functional relevance of the CXCR2 inhibitor reparixin in vivo, we determined its effects in two models of ALI, induced by either lipopolysaccharide (LPS) inhalation or acid instillation. In two ALI models in mice, we measured vascular permeability by Evans blue and evaluated neutrophil recruitment into the lung vasculature, interstitium and airspace by flow cytometry. Pharmacol. inhibition of CXCR2 by reparixin reduced CXCL1-induced leukocyte arrest in the microcirculation of the cremaster muscle, but did not influence arrest in response to leukotriene B4 (LTB4) demonstrating specificity. Reparixin (15 μg g-1) reduced neutrophil recruitment in the lung by approx. 50% in a model of LPS-induced ALI. A higher dose did not provide addnl. reduction of neutrophil recruitment. This dose also reduced accumulation of neutrophils in the interstitial compartment and vascular permeability in LPS-induced ALI. Furthermore, both prophylactic and therapeutic application of reparixin improved gas exchange, and reduced neutrophil recruitment and vascular permeability in a clin. relevant model of acid-induced ALI. Reparixin, a non-competitive allosteric CXCR2 inhibitor attenuates ALI by reducing neutrophil recruitment and vascular permeability.

REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 4 OF 23 CAPLUS
ACCESSION NUMBER: 2008:589691 CAPLUS
DOCUMENT NUMBER: 148:554109
TITLE: Method and use of nonionic polymers for increasing efficacy of anti-adhesive compositions in controlling inflammation and pain
INVENTOR(S): Chamness, Kathy L.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 15pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20080112921	A1	20080515	US 2006-598397	20061114
WO 2008063943	A2	20080529	WO 2007-US84387	20071112
WO 2008063943	A3	20090507		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI,

GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG,
KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME,
MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL,
PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN,
TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW,
GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,
BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA
PRIORITY APPLN. INFO.: US 2006-598397 A 20061114
AB The invention discloses a method and kits for increasing the efficiency of anti-adhesive compns. by parenterally administering a composition comprising an effective amount of at least one pharmaceutically acceptable anti-adhesive nonionic polymer to a site of injury, controlling inflammation at the site of injury, and reducing pain. The nonionic polymers are used with magnesium salts.

L3 ANSWER 5 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2008:412188 CAPLUS
DOCUMENT NUMBER: 148:394429
TITLE: CXC chemokine-mediated signaling targeting for treatment of a myelin disorder
INVENTOR(S): Miller, Robert H.; Padovani-Claudio, Dolly A.
PATENT ASSIGNEE(S): Case Western Reserve University, USA
SOURCE: PCT Int. Appl., 85pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2008039876	A1	20080403	WO 2007-US79602	20070926
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
CA 2664359	A1	20080403	CA 2007-2664359	20070926
US 20090041753	A1	20090212	US 2007-904634	20070926
EP 2066335	A1	20090610	EP 2007-843271	20070926
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, RS				

PRIORITY APPLN. INFO.: US 2006-847656P P 20060926
WO 2007-US79602 W 20070926
AB The invention discloses compns. and methods for targeting CXC chemokine-mediated signaling for treatment of a myelin disorder. The methodol. of the invention can be used to ameliorate neuropathies.
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 6 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2007:1075862 CAPLUS
DOCUMENT NUMBER: 147:541555
TITLE: A new and efficient method for the facile synthesis of N-acyl sulfonamides under Lewis acid catalysis
AUTHOR(S): Reddy, Chada Raji; Mahipal, Bodugam; Yaragorla, Srinivasa Rao
CORPORATE SOURCE: Organic Division-I, Indian Institute of Chemical Technology, Hyderabad, 500 007, India
SOURCE: Tetrahedron Letters (2007), 48(42), 7528-7532
CODEN: TELEAY; ISSN: 0040-4039
PUBLISHER: Elsevier Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 147:541555
AB The N-acylation of sulfonamides with carboxylic acid anhydrides in the presence of Lewis acids is described. Several Lewis acids such as BF3·Et2O, ZnCl2, MoCl5, TiCl4, B(C6F5)3, Sc(OTf)3 and I2 were found to catalyze the reaction efficiently to furnish the N-acylated products in good yields under solvent-free conditions. The reactions of various sulfonamides were studied with different carboxylic acid anhydrides including the less reactive benzoic and pivalic anhydrides, in the presence of 3 mol% ZnCl2 as the catalyst. Carboxylic acids were also successfully used as acylating agents via the mixed anhydride method.
OS.CITING REF COUNT: 5 THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)
REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 7 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2007:993886 CAPLUS
DOCUMENT NUMBER: 147:292200
TITLE: Methods and compositions for treating and preventing tumors
INVENTOR(S): Bonni, Azad M.; De la Iglesia, Nuria; Konopka, Genevieve
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 21pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 20070208074	A1	20070906	US 2007-657965		20070124
PRIORITY APPLN. INFO.:			US 2006-762033P	P	20060124

AB The present invention provides methods for reducing the growth or invasiveness of tumors.

L3 ANSWER 8 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2007:976827 CAPLUS
DOCUMENT NUMBER: 147:314799
TITLE: Reparixin, an inhibitor of CXCR2 function, attenuates inflammatory responses and promotes recovery of function after traumatic lesion to the spinal cord
AUTHOR(S): Gorio, Alfredo; Madaschi, Laura; Zadra, Giorgia; Marfia, Giovanni; Cavalieri, Barbara; Bertini, Riccardo; Di Giulio, Anna Maria
CORPORATE SOURCE: Pharmacological Laboratories, Department of Medicine, Surgery and Dentistry, Faculty of Medicine, University of Milan, Milan, Italy

SOURCE: Journal of Pharmacology and Experimental Therapeutics (2007), 322(3), 973-981
CODEN: JPETAB; ISSN: 0022-3565
PUBLISHER: American Society for Pharmacology and Experimental Therapeutics
DOCUMENT TYPE: Journal
LANGUAGE: English

AB It has been shown that the blockade of CXCR1 and CXCR2 receptors prevents ischemia/reperfusion damage in several types of vascular beds. Reparixin is a recently described inhibitor of human CXCR1/R2 and rat CXCR2 receptor activation. We applied reparixin in rats following traumatic spinal cord injury and determined therapeutic temporal and dosages windows. Treatment with reparixin significantly counteracts secondary degeneration by reducing oligodendrocyte apoptosis, migration to the injury site of neutrophils and ED-1-pos. cells. The observed preservation of the white matter might also be secondary to the enhanced proliferation of NG2-pos. cells. The expression of macrophage-inflammatory protein-2, tumor necrosis factor-α, interleukin (IL)-6, and IL-1β was also counteracted, and the proliferation of glial fibrillary acidic protein-pos. cells was markedly reduced. These effects resulted in a smaller post-traumatic cavity and in a significantly improved recovery of hind limb function. The best beneficial outcome of reparixin treatment required 7-day administration either by i.p. route (15 mg/kg) or s.c. infusion via osmotic pumps (10 mg/kg), reaching a steady blood level of 8 μg/mL. Methylprednisolone was used as a reference drug; such treatment reduced cytokine production but failed to affect the rate of hind limb recovery.

OS.CITING REF COUNT: 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)
REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 9 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2007:807542 CAPLUS
DOCUMENT NUMBER: 147:314717
TITLE: The interleukin-8 (IL-8/CXCL8) receptor inhibitor reparixin improves neurological deficits and reduces long-term inflammation in permanent and transient cerebral ischemia in rats
AUTHOR(S): Villa, Pia; Triulzi, Sara; Cavalieri, Barbara; Di Bitondo, Rosa; Bertini, Riccardo; Barbera, Sara; Bigini, Paolo; Mennini, Tiziana; Gelosa, Paolo; Tremoli, Elena; Sironi, Luigi; Ghezzi, Pietro
CORPORATE SOURCE: Mario Negri Institute, Milan, 20157, Italy
SOURCE: Molecular Medicine (Manhasset, NY, United States) (2007), 13(3-4), 125-133
CODEN: MOMEF3; ISSN: 1076-1551
PUBLISHER: Feinstein Institute for Medical Research
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Leukocyte infiltration is viewed as a pharmacol. target in cerebral ischemia. We previously reported that reparixin, a CXCL8 receptor blacker that inhibits neutrophil infiltration, and related mols. can reduce infarct size in a rat model of transient middle cerebral artery occlusion (MCAO). The study aims were to compare the effects of reparixin in transient and permanent MCAO using varied treatment schedules and therapeutic windows to evaluate effects on long-term neurol. deficits and late inflammatory response. Reparixin, administered for 1 to 3 days, 3.5 to 6 h after MCAO, ameliorates neurol. function recovery and inhibits long-term inflammation. The infarct size reduction at 24 h, evaluated by TTC staining, is more pronounced in transient MCAO. MRI anal. identified a decrease in the progression of infarct size by reparixin that was more

evident at 48 h in permanent MCAO, and was associated with a significantly improved recovery from long-term neurol. deficits.

OS.CITING REF COUNT: 5 THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)

REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 10 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2007:451972 CAPLUS

DOCUMENT NUMBER: 147:109107

TITLE: Reparixin, a specific interleukin-8 inhibitor, has no effects on inflammation during endotoxemia

AUTHOR(S): Leitner, J. M.; Mayr, F. B.; Firbas, C.; Spiel, A. O.; Steinlechner, B.; Novellini, R.; Jilma, B.

CORPORATE SOURCE: Department of Clinical Pharmacology, Division of Immunohaematology, Medical University of Vienna, Austria

SOURCE: International Journal of Immunopathology and Pharmacology (2007), 20(1), 25-36
CODEN: IJIPE4; ISSN: 0394-6320

PUBLISHER: Biolife s.a.s.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Reparixin antagonizes interleukin-8 (IL-8) on the level of signal transduction in vitro. We hypothesized that IL-8 mediates some of the reactions occurring during acute inflammation and specifically that IL-8 may be a mediator of endotoxin induced neutrophilia. We therefore tested the effects of reparixin on humoral and cellular parameters in LPS-induced acute systemic inflammation. The study is a randomized (3:2 active:placebo), double-blind, placebo-controlled parallel group trial. Twenty healthy male volunteers randomly received either reparixin (12) or placebo (8) i.v. One hour after the start of reparixin/placebo infusion a bolus of 2 ng/kg endotoxin was infused over 1-2 min. Blood samples were obtained over 24 h. Reparixin, being metabolized to ibuprofen, suppressed serum thromboxane B2 levels by 78% compared to baseline and control at 8 h. LPS-induced neutrophilia was not significantly affected by reparixin in human volunteers. Consistently, reparixin did not alter the lymphocyte or monocyte counts and had no effect on LPS-induced systemic inflammation as measured by tumor necrosis factor alpha (TNF-α) or interleukin-6 (IL-6) release. Regulation of IL-8 receptors CXCR1 and 2 and the degranulation marker CD11b showed the expected kinetics. Reparixin had no effect on thrombin formation as measured by prothrombin fragment (F1+2). In conclusion, our study showed that reparixin was safe but had no impact on endotoxin induced inflammation. In contrast to previous studies with its metabolite ibuprofen, reparixin does not enhance inflammation in this model.

OS.CITING REF COUNT: 5 THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)

REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 11 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2006:704377 CAPLUS

DOCUMENT NUMBER: 145:369213

TITLE: Species differences in the pharmacokinetics and metabolism of Reparixin in rat and dog

AUTHOR(S): Midgley, I.; Fitzpatrick, K.; Wright, S. J.; John, B. A.; Peard, A. J.; Major, R. M.; Holding, J. D.; McBurney, A.; Anacardio, R.; Novellini, R.; Ferrari, M. P.

CORPORATE SOURCE: Department of Drug Metabolism, Huntingdon Life Sciences Ltd, Huntingdon, UK

SOURCE: Xenobiotica (2006), 36(5), 419-440
CODEN: XENOBH; ISSN: 0049-8254
PUBLISHER: Taylor & Francis Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The pharmacokinetics and metabolism of Reparixin (formerly Repertaxin), a potent and specific inhibitor of the chemokine CXCL8, were investigated in rats and dogs after i.v. administration of [14C]Reparixin L-lysine salt. Protein binding of Reparixin was investigated in vitro in rat, dog, rabbit, cynomolgus monkey, and human plasma. Plasma protein binding of Reparixin was >99% in the laboratory animals and humans up to 50 μg mL-1, but lower at higher concns. Although radioactivity was rapidly distributed into rat tissues, Vss was low (.apprx.0.15 L kg-1) in both rat and dog. Nevertheless, Reparixin was more rapidly eliminated in rats (t1/2 .apprx.0.5 h) than in dogs (t1/2 .apprx.10 h). Systemic exposure in dog was due primarily to parent drug, but metabolites played a more prominent role in rat. Oxidation of the iso-Bu side-chain was the major metabolic pathway in rat, whereas hydrolysis of the amide bond predominated in dog. Urinary excretion, which accounted for 80-82% of the radioactive dose, was the major route of elimination in both species, and biotransformation of Reparixin was complete before excretion.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 12 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2006:608541 CAPLUS
DOCUMENT NUMBER: 145:62689
TITLE: Preparation of 2-arylpropionamides for the inhibition of the chemotactic activation induced by C5a
INVENTOR(S): Allegretti, Marcello; Bertini, Riccardo; Beccari, Andrea; Moriconi, Alessio; Aramini, Andrea; Bizzarri, Cinzia; Colotta, Francesco
PATENT ASSIGNEE(S): Dompe' S.p.A., Italy
SOURCE: PCT Int. Appl., 51 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

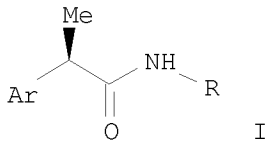
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006063999	A1	20060622	WO 2005-EP56742	20051213
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
AU 2005315591	A1	20060622	AU 2005-315591	20051213
CA 2589495	A1	20060622	CA 2005-2589495	20051213
EP 1856031	A1	20071121	EP 2005-817430	20051213
EP 1856031	B1	20090225		
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,				

IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU

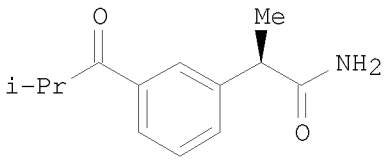
JP 2008524157	T	20080710	JP 2007-546040		20051213
AT 423760	T	20090315	AT 2005-817430		20051213
ES 2322487	T3	20090622	ES 2005-817430		20051213
MX 2007007133	A	20070808	MX 2007-7133		20070614
US 20080312293	A1	20081218	US 2007-721971		20070615
KR 2007112365	A	20071123	KR 2007-715497		20070706
NO 2007003622	A	20070917	NO 2007-3622		20070713
CN 101184726	A	20080521	CN 2005-80048026		20070810
PRIORITY APPLN. INFO.:			EP 2004-29684	A	20041215
			WO 2005-EP56742	W	20051213

OTHER SOURCE(S): CASREACT 145:62689; MARPAT 145:62689

GI



I



II

AB Title compds. I [Ar = Ph substituted in the meta position by a group selected from alkanoyl, cycloalkanoyl, heteroarylcarbonyl, etc.; R = H, OH, alkyl, etc.] were prepared For example, chlorination of (R)-2-(3-isobutyrylphenyl)propionic acid, e.g., prepared from 2-[(3-carboxy)phenyl]propionitrile in 3 steps, using thionyl chloride followed by treatment with ammonia afforded compound II. In C5a induced PMNs chemotaxis inhibition assays, compound II exhibited the activity of 50 ± 7% at 10-7 M. Compds. I are claimed useful for the treatment of sepsis, psoriasis, etc.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 13 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2005:1301378 CAPLUS

DOCUMENT NUMBER: 144:324102

TITLE: Neutrophil recruitment in the reperfused-injured rat liver was effectively attenuated by repertaxin, a novel allosteric non-competitive inhibitor of CXCL8 receptors: A therapeutic approach for the treatment of post-ischemic hepatic syndromes

AUTHOR(S): Cavalieri, B.; Mosca, M.; Ramadori, P.; Perrelli, M.-G.; De Simone, L.; Colotta, F.; Bertini, R.; Poli, G.; Cutrin, J. C.

CORPORATE SOURCE: Laboratory of Experimental Liver Pathology, Department of Clinical and Biological Sciences, University of Turin, L'Aquila, Italy
SOURCE: International Journal of Immunopathology and Pharmacology (2005), 18(3), 475-486
CODEN: IJIPE4; ISSN: 0394-6320
PUBLISHER: Biolife s.a.s.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Hepatic reperfusion injury represents a crucial problem in several clin. situations including liver transplantation, extensive hepatectomy and hypovolemic shock with resuscitation. Repertaxin is a new non-competitive allosteric blocker of interleukin-8 (CXCL8) receptors, which by locking CXCR1/R2 in an inactive conformation, prevents receptor signaling and polymorphonuclear leukocyte (PMN) chemotaxis. The present study shows that repertaxin dramatically prevents rat post-ischemic hepatocellular necrosis (80% of inhibition) and PMN infiltration (96% of inhibition) at a clin.-relevant time (24 h) of reperfusion. Treatment with repertaxin by continuous infusion is demonstrated to be the optimal route of administration of the compound especially in view of its clin. therapeutic use. Because repertaxin has proven to be safe and well tolerated in different animal studies and in phase I studies in human volunteers, it is in fact a candidate novel therapeutic agent for the prevention and treatment of hepatic post-ischemic injury.
OS.CITING REF COUNT: 13 THERE ARE 13 CAPLUS RECORDS THAT CITE THIS RECORD (13 CITINGS)
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 14 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2005:460353 CAPLUS
DOCUMENT NUMBER: 143:145782
TITLE: 2-Arylpropionic CXC Chemokine Receptor 1 (CXCR1) Ligands as Novel Noncompetitive CXCL8 Inhibitors
AUTHOR(S): Allegretti, Marcello; Bertini, Riccardo; Cesta, Maria Candida; Bizzarri, Cinzia; Di Bitondo, Rosa; Di Cioccio, Vito; Galliera, Emanuela; Berdini, Valerio; Topai, Alessandra; Zampella, Giuseppe; Russo, Vincenzo; Di Bello, Nicoletta; Nano, Giuseppe; Nicolini, Luca; Locati, Massimo; Fantucci, Piercarlo; Florio, Saverio; Colotta, Francesco
CORPORATE SOURCE: Dompe Research and Development, Dompe S.p.A., L'Aquila, 67100, Italy
SOURCE: Journal of Medicinal Chemistry (2005), 48(13), 4312-4331
CODEN: JMCMAR; ISSN: 0022-2623
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 143:145782
AB The CXC chemokine CXCL8/IL-8 plays a major role in the activation and recruitment of polymorphonuclear (PMN) cells at inflammatory sites. CXCL8 activates PMNs by binding the seven-transmembrane (7-TM) G-protein-coupled receptors CXC chemokine receptor 1 (CXCR1) and CXC chemokine receptor 2 (CXCR2). (R)-Ketoprofen (1) was previously reported to be a potent and specific noncompetitive inhibitor of CXCL8-induced human PMNs chemotaxis. The authors report here mol. modeling studies showing a putative interaction site of 1 in the TM region of CXCR1. The binding model was confirmed by alanine scanning mutagenesis and photoaffinity labeling expts. The mol. model driven medicinal chemical optimization of 1 led to a new class of potent and specific inhibitors of CXCL8 biol. activity. Among these, repertaxin was selected as a clin. candidate drug for

prevention of postischemia reperfusion injury.
OS.CITING REF COUNT: 22 THERE ARE 22 CAPLUS RECORDS THAT CITE THIS RECORD (23 CITINGS)
REFERENCE COUNT: 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 15 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2005:437986 CAPLUS
DOCUMENT NUMBER: 143:53210
TITLE: Inhibition of the chemokine receptor CXCR2 prevents kidney graft function deterioration due to ischemia/reperfusion
AUTHOR(S): Cugini, Daniela; Azzollini, Nadia; Gagliardini, Elena; Cassis, Paola; Bertini, Riccardo; Colotta, Francesco; Noris, Marina; Remuzzi, Giuseppe; Benigni, Ariela
CORPORATE SOURCE: Transplant Research Center "Chiara Cucchi de Alessandri e Gilberto Crespi" Mario Negri Institute for Pharmacological Research, Bergamo, Italy
SOURCE: Kidney International (2005), 67(5), 1753-1761
CODEN: KDYIA5; ISSN: 0085-2538
PUBLISHER: Blackwell Publishing, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Background: Ischemia/reperfusion (I/R) injury after organ transplantation is a major cause of delayed graft function. Following I/R, locally produced CXC chemokines attract and activate granulocytes, which in turn promote graft damage. Methods: We examined the involvement of granulocyte recruitment via the CXCR2 pathway in a rat model of 4 h cold ischemia followed by kidney transplantation. Serum creatinine and intragraft granulocyte infiltration were monitored in the early phase posttransplant. A CXCR2 inhibitor, repertaxin, was given to recipients before transplantation (at -24 h or -8 h or -2 h), immediately before reperfusion and 2 h later. Results: An increase of granulocyte chemoattractant CINC-1/interleukin-8 (IL-8) mRNA expression after I/R both in syngeneic and allogeneic transplantation was associated with a marked infiltration of granulocytes in renal tissue. In syngeneic transplantation, Lewis rats given 15 mg/kg repertaxin 24 h before surgery had granulocyte graft infiltration and serum creatinine levels significantly reduced in respect to vehicle-treated animals. Intermediate effects were observed with 5 mg/kg, whereas the dose of 30 mg/kg had toxic effects. We found that reducing the pretreatment time to 8 h before surgery was still effective. Prevention of granulocyte infiltration and serum creatinine increase was also obtained in allogeneic transplantation, when Brown Norway recipients of Lewis kidneys were given 15 mg/kg repertaxin starting 8 h before surgery. Conclusion: Repertaxin treatment of the recipient animal was effective in preventing granulocyte infiltration and renal function impairment both in syngeneic and in allogeneic settings. The possibility to modulate I/R injury in this rat model opens new perspectives for preventing posttransplant delayed graft function in humans.
OS.CITING REF COUNT: 33 THERE ARE 33 CAPLUS RECORDS THAT CITE THIS RECORD (33 CITINGS)
REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 16 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2005:319144 CAPLUS
DOCUMENT NUMBER: 142:475974
TITLE: Neuroprotection with the CXCL8 inhibitor repertaxin in transient brain ischemia
AUTHOR(S): Garau, Angela; Bertini, Riccardo; Colotta, Francesco; Casilli, Federica; Bigini, Paolo; Cagnotto, Alfredo; Mennini, Tiziana; Ghezzi, Pietro; Villa, Pia

CORPORATE SOURCE: "Mario Negri" Institute for Pharmacological Research, Milan, Italy
SOURCE: Cytokine+ (2005), 30(3), 125-131
CODEN: CYTIE9; ISSN: 1043-4666
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Infiltration of polymorphonuclear neutrophils (PMNs) is thought to play a role in ischemic brain damage. The present study investigated the effect of repertaxin, a new noncompetitive allosteric inhibitor for the receptors of the inflammatory chemokine CXC ligand 8 (CXCL8)/interleukin-8 (IL-8), on PMN infiltration and tissue injury in rats. Cerebral ischemia was induced by permanent or transient occlusion of the middle cerebral artery and myeloperoxidase activity, a marker of PMN infiltration, and infarct volume were evaluated 24 h later. Repertaxin (15 mg/kg) was administered systemically at the time of ischemia and every 2 h for four times. In permanent ischemia repertaxin reduced PMN infiltration by 40% in the brain cortex but did not limit tissue damage. In transient ischemia (90-min ischemia followed by reperfusion), repertaxin inhibited PMN infiltration by 54% and gave 44% protection from tissue damage. Repertaxin had anti-inflammatory and neuroprotective effects also when given at reperfusion and even at 2 h of reperfusion. The protective effect of repertaxin did not interfere with brain levels of the chemokine. Since the PMN infiltration and its inhibition by repertaxin were comparable in the two models we conclude that reperfusion induces PMN activation, and inhibition of CXCL8 by repertaxin might be of pharmacol. interest in transient ischemia.

OS.CITING REF COUNT: 19 THERE ARE 19 CAPLUS RECORDS THAT CITE THIS RECORD (19 CITINGS)
REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 17 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2005:201863 CAPLUS
DOCUMENT NUMBER: 142:385080
TITLE: Predicting Human Serum Albumin Affinity of Interleukin-8 (CXCL8) Inhibitors by 3D-QSPR Approach
AUTHOR(S): Aureli, Loretta; Cruciani, Gabriele; Cesta, Maria Candida; Anacardio, Roberto; De Simone, Lucio; Moriconi, Alessio
CORPORATE SOURCE: Molecular Discovery Ltd., London, W1A 3BQ, UK
SOURCE: Journal of Medicinal Chemistry (2005), 48(7), 2469-2479
CODEN: JMCMAR; ISSN: 0022-2623
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 142:385080

AB A novel class of 2-(R)-phenylpropionamides has been recently reported to inhibit in vitro and in vivo interleukin-8 (CXCL8)-induced biol. activities. These CXCL8 inhibitors are derivs. of phenylpropionic nonsteroidal antiinflammatory drugs (NSAIDs), high-affinity ligands for site II of human serum albumin (HSA). Up to date, only a limited number of in silico models for the prediction of albumin protein binding are available. A three-dimensional quant. structure-property relationship (3D-QSPR) approach was used to model the exptl. affinity constant (Ki) to plasma proteins of 37 structurally related mols., using physicochem. and 3D-pharmacophoric descriptors. Mol. docking studies highlighted that training set mols. preferentially bind site II of HSA. The obtained model shows satisfactory statistical parameters both in fitting and predicting validation. External validation confirmed the statistical significance of the chemometric model, which is a powerful tool for the prediction of HSA

binding in virtual libraries of structurally related compds.
OS.CITING REF COUNT: 14 THERE ARE 14 CAPLUS RECORDS THAT CITE THIS RECORD (14 CITINGS)
REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 18 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2005:28032 CAPLUS
DOCUMENT NUMBER: 142:190637
TITLE: Inhibition of interleukin-8 (CXCL8/IL-8) responses by repertaxin, a new inhibitor of the chemokine receptors CXCR1 and CXCR2
AUTHOR(S): Casilli, Federica; Bianchini, Andrea; Gloaguen, Isabelle; Biordi, Leda; Alesse, Edoardo; Festuccia, Claudio; Cavalieri, Barbara; Strippoli, Raffaele; Cervellera, Maria Neve; Di Bitondo, Rosa; Ferretti, Elisabetta; Mainiero, Fabrizio; Bizzarri, Cinzia; Colotta, Francesco; Bertini, Riccardo
CORPORATE SOURCE: Dompe S.p.A. Research Center, L'Aquila, Italy
SOURCE: Biochemical Pharmacology (2005), 69(3), 385-394
CODEN: BCPCA6; ISSN: 0006-2952
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Repertaxin is a new non-competitive allosteric blocker of interleukin-8 (CXCL8/IL-8) receptors (CXCR1/R2), which by locking CXCR1/R2 in an inactive conformation prevents receptor signaling and human polymorphonuclear leukocyte (PMN) chemotaxis. Given the unique mode of action of repertaxin it was important to examine the ability of repertaxin to inhibit a wide range of biol. activities induced by CXCL8 in human leukocytes. Our results show that repertaxin potently and selectively blocked PMN adhesion to fibrinogen and CD11b up-regulation induced by CXCL8. Reduction of CXCL8-mediated PMN adhesion by repertaxin was paralleled by inhibition of PMN activation including secondary and tertiary granule release and pro-inflammatory cytokine production, whereas PMN phagocytosis of Escherichia coli bacteria was unaffected. Repertaxin also selectively blocked CXCL8-induced T lymphocyte and natural killer (NK) cell migration. These data suggest that repertaxin is a potent and specific inhibitor of a wide range of CXCL8-mediated activities related to leukocyte recruitment and functional activation in inflammatory sites.
OS.CITING REF COUNT: 22 THERE ARE 22 CAPLUS RECORDS THAT CITE THIS RECORD (22 CITINGS)
REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 19 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2004:803495 CAPLUS
DOCUMENT NUMBER: 141:343217
TITLE: Repertaxin, a novel inhibitor of rat CXCR2 function, inhibits inflammatory responses that follow intestinal ischaemia and reperfusion injury
AUTHOR(S): Souza, Danielle G.; Bertini, Riccardo; Vieira, Angelica T.; Cunha, Fernando Q.; Poole, Steve; Allegretti, Marcello; Colotta, Francesco; Teixeira, Mauro M.
CORPORATE SOURCE: Immunopharmacology, Departamento de Bioquimica e Imunologia, ICB, Universidade Federal de Minas Gerais, Belo Horizonte, Brazil
SOURCE: British Journal of Pharmacology (2004), 143(1), 132-142
CODEN: BJPCBM; ISSN: 0007-1188
PUBLISHER: Nature Publishing Group

DOCUMENT TYPE: Journal
LANGUAGE: English
AB Neutrophils are thought to play a major role in the mediation of reperfusion injury. CXC chemokines are known inducers of neutrophil recruitment. Here, we assessed the effects of Repertaxin, a novel low mol. weight inhibitor of human CXCL8 receptor activation, on the local, remote and systemic injuries following intestinal ischemia and reperfusion (I/R) in the rat. Pre-incubation of rat neutrophils with Repertaxin (10-11-10-6 M) inhibited the chemotaxis of neutrophils induced by human CXCL8 or rat CINC-1, but not that induced by fMLP, PAF or LTB4, in a concentration-dependent manner. Repertaxin also prevented CXCL8-induced calcium influx but not CXCL8 binding to purified rat neutrophils. In a model of mild I/R injury (30 min of ischemia and 30 min of reperfusion), Repertaxin dose-dependently (3-30 mg kg-1) inhibited the increase in vascular permeability and neutrophil influx. Maximal inhibition occurred at 30 mg kg-1. Following severe I/R injury (120 min of ischemia and 120 min of reperfusion), Repertaxin (30 mg kg-1) markedly prevented neutrophil influx, the increase in vascular permeability both in the intestine and the lungs. Moreover, there was prevention of hemorrhage in the intestine of reperfused animals. Repertaxin effectively suppressed the increase in tissue (intestine and lungs) and serum concns. of TNF-α and the reperfusion-associated lethality. For comparison, we also evaluated the effects of an anti-CINC-1 antibody in the model of severe I/R injury. Overall, the antibody effectively prevented tissue injury, systemic inflammation and lethality. However, the effects of the antibody were in general of lower magnitude than those of Repertaxin. In conclusion, CINC-1 and possibly other CXC chemokines, acting on CXCR2, have an important role during I/R injury. Thus, drugs, such as Repertaxin, developed to block the function of the CXCR2 receptor may be effective at preventing reperfusion injury in relevant clin. situations.
OS.CITING REF COUNT: 38 THERE ARE 38 CAPLUS RECORDS THAT CITE THIS RECORD (38 CITINGS)
REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 20 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2004:703810 CAPLUS
DOCUMENT NUMBER: 141:343408
TITLE: Noncompetitive allosteric inhibitors of the inflammatory chemokine receptors CXCR1 and CXCR2: Prevention of reperfusion injury
AUTHOR(S): Bertini, Riccardo; Allegretti, Marcello; Bizzarri, Cinzia; Moriconi, Alessio; Locati, Massimo; Zampella, Giuseppe; Cervellera, Maria N.; di Cioccio, Vito; Cesta, Maria C.; Galliera, Emanuela; Martinez, Fernando O.; di Bitondo, Rosa; Troiani, Giulia; Sabbatini, Vilma; D'Anniballe, Gaetano; Anacardio, Roberto; Cutrin, Juan C.; Cavalieri, Barbara; Mainiero, Fabrizio; Strippoli, Raffaele; Villa, Pia; di Girolamo, Maria; Martin, Franck; Gentile, Marco; Santoni, Angela; Corda, Daniela; Poli, Giuseppe; Mantovani, Alberto; Ghezzi, Pietro; Colotta, Francesco
CORPORATE SOURCE: Dompe, L'Aquila, 67100, Italy
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2004), 101(32), 11791-11796
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The chemokine CXC ligand 8 (CXCL8)/IL-8 and related agonists recruit and activate polymorphonuclear cells by binding the CXC chemokine receptor 1

(CXCR1) and CXCR2. Here the authors characterize the unique mode of action of a small-mol. inhibitor (repertaxin) of CXCR1 and CXCR2. Structural and biochem. data are consistent with a noncompetitive allosteric mode of interaction between CXCR1 and repertaxin, which, by locking CXCR1 in an inactive conformation, prevents signaling. Repertaxin is an effective inhibitor of polymorphonuclear cell recruitment in vivo and protects organs against reperfusion injury. Targeting the repertaxin interaction site of CXCR1 represents a general strategy to modulate the activity of chemoattractant receptors.

OS.CITING REF COUNT: 75 THERE ARE 75 CAPLUS RECORDS THAT CITE THIS RECORD (75 CITINGS)

REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 21 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2004:498365 CAPLUS

DOCUMENT NUMBER: 141:173953

TITLE: Acylmethanesulfonamides as new acylating agents for primary amines

AUTHOR(S): Coniglio, Silvia; Aramini, Andrea; Cesta, M. Candida; Colagioia, Sandro; Curti, Roberto; D'Alessandro, Fabrizio; D'Anniballe, Gaetano; D'Elia, Valerio; Nano, Giuseppe; Orlando, Valerie; Allegretti, Marcello

CORPORATE SOURCE: Dompe Research and Development, Chemistry Department, Dompe S.p.A., L'Aquila, 67100, Italy

SOURCE: Tetrahedron Letters (2004), 45(28), 5375-5378
CODEN: TELEAY; ISSN: 0040-4039

PUBLISHER: Elsevier

DOCUMENT TYPE: Journal

LANGUAGE: English

OTHER SOURCE(S): CASREACT 141:173953

AB A simple and efficient procedure for the preparation of secondary amides through internal condensation of acylmethanesulfonamides ammonium salts is described. The selective acylation of mixed primary-secondary amines could be an attractive application of this method.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 22 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2002:615394 CAPLUS

DOCUMENT NUMBER: 137:150277

TITLE: Use of (R)-ibuprofen methanesulfonamide and salts thereof in the treatment and prevention of ischemia/reperfusion injury or rejection reactions of transplanted organs

INVENTOR(S): Bertini, Riccardo; Colotta, Francesco; Novellini, Roberto

PATENT ASSIGNEE(S): Dompe S.p.A., Italy

SOURCE: PCT Int. Appl., 16 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002062330	A2	20020815	WO 2002-EP946	20020130
WO 2002062330	A3	20030403		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,

CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT,
RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US,
UZ, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB,
GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA,
GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA 2432432	A1	20020815	CA 2002-2432432	20020130
CA 2432432	C	20080325		
AU 2002250869	A1	20020819	AU 2002-250869	20020130
AU 2002250869	B2	20061019		
EE 200300340	A	20031015	EE 2003-340	20020130
EP 1355641	A2	20031029	EP 2002-719742	20020130
EP 1355641	B1	20050921		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
HU 2003003024	A2	20031229	HU 2003-3024	20020130
HU 2003003024	A3	20050728		
BR 2002006804	A	20040203	BR 2002-6804	20020130
JP 2004517948	T	20040617	JP 2002-562337	20020130
CN 1561205	A	20050105	CN 2002-804226	20020130
NZ 526655	A	20050225	NZ 2002-526655	20020130
RU 2257895	C2	20050810	RU 2003-126600	20020130
AT 304846	T	20051015	AT 2002-719742	20020130
ES 2248541	T3	20060316	ES 2002-719742	20020130
IL 157180	A	20090615	IL 2002-157180	20020130
ZA 2003004861	A	20040630	ZA 2003-4861	20030623
NO 2003003273	A	20030718	NO 2003-3273	20030718
KR 857898	B1	20080910	KR 2003-709714	20030723
MX 2003006686	A	20040531	MX 2003-6686	20030725
US 20040102520	A1	20040527	US 2003-250465	20031002
US 7560487	B2	20090714		
PRIORITY APPLN. INFO.:			IT 2001-MI206 A	20010202
			WO 2002-EP946 W	20020130

AB The use of (R)-ibuprofen methanesulfonamide is described for the preparation of medicaments for the treatment and prevention of ischemia/reperfusion injury or functional injury resulting from rejection reactions of transplanted organs. In particular, the use of non-toxic salts of (R)-ibuprofen methanesulfonamide, such as the (L)-lysine salt (DF 1681B), is described for the prevention and the treatment of rejection reactions of transplanted kidneys. DF 1681B prevented renal function impairment secondary to cold ischemia in a rat model of kidney transplantation.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 23 OF 23 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2000:290989 CAPLUS
DOCUMENT NUMBER: 132:321722
TITLE: Preparation of N-(2-arylpropionyl)sulfonamides as inhibitors of neutrophil chemotaxis and degranulation induced by interleukin 8.
INVENTOR(S): Bertini, Riccardo; Bizzarri, Cinzia; Sabbatini, Vilma; Porzio, Stefano; Caselli, Gianfranco; Allegretti, Marcello; Cesta, Maria Candida; Gandolfi, Carmelo A.; Mantovanini, Marco; Colotta, Francesco
PATENT ASSIGNEE(S): Dompe' S.P.A., Italy; et al.
SOURCE: PCT Int. Appl., 41 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000024710	A1	20000504	WO 1999-EP7740	19991014
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
IT 1303249	B1	20001106	IT 1998-MI2280	19981023
CA 2347752	A1	20000504	CA 1999-2347752	19991014
BR 9914741	A	20010703	BR 1999-14741	19991014
EP 1123276	A1	20010816	EP 1999-953824	19991014
EP 1123276	B1	20030108		
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TR 200101124	T2	20011022	TR 2001-1124	19991014
HU 2001003793	A2	20020328	HU 2001-3793	19991014
HU 2001003793	A3	20030929		
HU 225107	B1	20060628		
EE 200100233	A	20020815	EE 2001-233	19991014
EE 4912	B1	20071015		
JP 2002528434	T	20020903	JP 2000-578281	19991014
JP 4194761	B2	20081210		
AT 230723	T	20030115	AT 1999-953824	19991014
ES 2190264	T3	20030716	ES 1999-953824	19991014
NZ 511077	A	20030829	NZ 1999-511077	19991014
AU 769850	B2	20040205	AU 2000-10375	19991014
CN 1615833	A	20050518	CN 2004-10085635	19991014
RU 2255084	C2	20050627	RU 2001-113733	19991014
IL 142496	A	20060115	IL 1999-142496	19991014
CZ 296434	B6	20060315	CZ 2001-1441	19991014
CN 100368394	C	20080213	CN 1999-812451	19991014
SK 286372	B6	20080805	SK 2001-538	19991014
MX 2001003987	A	20020225	MX 2001-3987	20010420
NO 2001002000	A	20010620	NO 2001-2000	20010423
US 6887903	B1	20050503	US 2001-830075	20011121
HK 1041255	A1	20080718	HK 2002-102780	20020412
NZ 525084	A	20040827	NZ 2003-525084	20030401
US 20030216392	A1	20031120	US 2003-460203	20030613
US 6881755	B2	20050419		
AU 2003259648	A1	20031127	AU 2003-259648	20031103
AU 2003259648	B2	20060525		
EP 1579859	A1	20050928	EP 2004-7177	20040325
EP 1579859	B1	20061213		
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AT 347883	T	20070115	AT 2004-7177	20040325
ES 2279248	T3	20070816	ES 2004-7177	20040325
AU 2005226901	A1	20051006	AU 2005-226901	20050317
CA 2555162	A1	20051006	CA 2005-2555162	20050317
WO 2005092315	A1	20051006	WO 2005-EP2822	20050317
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            NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM,
            SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
        RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
            AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
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            RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML,
            MR, NE, SN, TD, TG
     CN 1933825           A     20070321    CN 2005-80009560       20050317
     BR 2005009167        A     20070911    BR 2005-9167           20050317
     JP 2007530478        T     20071101    JP 2007-504310         20050317
     MX 2006009085        A     20070402    MX 2006-9085           20060810
     KR 2007018015        A     20070213    KR 2006-718031         20060905
     NO 2006004793        A     20061023    NO 2006-4793           20061023
     US 20090030083       A1    20090129    US 2006-588454         20061205
PRIORITY APPLN. INFO.:                      IT 1998-MI2280      A  19981023
                                            AU 2000-10375       A3 19991014
                                            WO 1999-EP7740      W  19991014
                                            US 2001-830075      A3 20011121
                                            EP 2004-7177        A  20040325
                                            WO 2005-EP2822      W  20050317

OTHER SOURCE(S):         MARPAT 132:321722
AB   R2CHMeCONR1SO2R (R2 = aryl; R = alkyl, CF3, cyclohexyl, o-tolyl,
     3-pyridyl, 2-pyridylethyl, p-cyanophenylmethyl, p-aminophenylmethyl,
     3-cyano-1-Pr, 4-aminobutyl, etc.; R1 = H, alkyl), were prepared  Thus,
     (R)-2-(4-isobutylphenyl)propionyl chloride in MeCN was added to NH3 in H2O
     at 0-5° to give (R)-2-(4-isobutylphenyl)propionamide.  Title
     compds. inhibited chemotaxis of PMN human leukocytes with IC50 = 10-7 to
     10-9M.
OS.CITING REF COUNT:     13    THERE ARE 13 CAPLUS RECORDS THAT CITE THIS
                               RECORD (13 CITINGS)
REFERENCE COUNT:         5     THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT


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---Logging off of STN---



Connection closed by remote host
END

 Unable to generate the STN prompt.
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